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# Diathesis—Stress Theories in the Context of Life Stress Research

## Implications for the Depressive Disorders

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**ABSTRACT**

Advances in the conceptualization and measurement of life stress in the past 2 decades raise several questions concerning traditional diathesis—stress theories of psychopathology. First, comprehensive measures of life stress force investigators to become more precise about the particular stressful circumstances hypothesized to interact with diatheses. Second, the influence of the diathesis on a person's life is typically ignored, which results in several types of possible bias in the assessment of life stress. Finally, information is available on diatheses and stress for specific disorders to provide a foundation for more empirically based hypotheses about diathesis—stress interactions. This possibility is outlined for depression. Such an approach provides the basis for developing broader, yet more specific, frameworks for investigating diathesis—stress theories of psychopathology in general and of depression in particular.

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In attempting to understand the antecedents of psychopathology, theorists historically have sought explanations from two spheres. On the one hand, the belief has long been held that people who develop a psychiatric disorder differ premorbidly from those who do not. Such differences were thought to be constitutional in origin. Well over 100 years ago, for example, the terminology of a diathesis for mental disease was quite active in the psychiatric vocabulary (e.g., Beard, 1881 ). [1] On the other hand, the belief also has long been held that stress is an important factor in the development of psychological disturbances ( Hawkes, 1857 ; Hinkle, 1977 ; Rees, 1976 ; Rosen, 1959 ). Yet, it has been recognized that not all people, even when exposed to the most dire of environmental conditions, necessarily break down ( B. P. Dohrenwend & Dohrenwend, 1979 ; Grinker & Spiegel, 1963 ). Among the first to bring these two spheres together in a more unified fashion ( Meehl, 1962 ) and to develop the specific terminology of diathesis—stress interactions ( Bleuler, 1963 ; Rosenthal, 1963 ) were theories of schizophrenia proposed during the 1960s. Most recently, theories of depression have explicitly adopted such models (e.g., Abramson, Metalsky, & Alloy, 1989 ; Bebbington, 1987 ; Beck, 1987 ; McGuffin, Katz, & Bebbington, 1988 ; Robins & Block, 1989 ). The basic premise is that stress activates a diathesis, transforming the potential of predisposition into the presence of psychopathology.

Recent efforts to incorporate diathesis—stress premises in theories of depression appear promising. However, invoking new domains of predisposition other than constitutional (e.g., cognitive or social vulnerability) and applying them to other forms of psychopathology (e.g., depression) require a reevaluation of several premises involving diathesis—stress interactions. Three topics consequently constitute the foci of the present article. First, over the past 20 years, the conceptualization and measurement of life stress have become

more detailed and sophisticated. Definitions and operational procedures adopted by different investigators vary in terms of the qualities and dimensions of experience that are included, the importance assigned to these qualities and dimensions, and the manner in which these different components of stress are combined. Ironically, depending on how these advances are used, they may obscure rather than reveal the nature of specific diathesis—stress interactions for particular disorders.

The second topic concerns the diathesis and its implications for stress. As noted previously, early formulations of the diathesis—stress model were predicated on biological factors (e.g., Beard, 1881 ; Meehl, 1962 ). Owing to the inferred temporal precedence and assumed quiescent nature of the diathesis in the developmental scheme, the interpretation of a significant interaction seemed straightforward: Stress activated the diathesis, which in turn brought about onset of disorder. The complementary influence, that of the diathesis on stress, was typically ignored. The recent incarnations of the diathesis—stress model for depression have suggested that other domains of predisposition can be conceptualized as a diathesis (e.g., cognitive or social factors; Abramson et al., 1989 ; Alloy, Hartlage, & Abramson, 1988 ; Bebbington, 1987 ; Beck, 1987 ; Brown & Harris, 1978 ; Perris, 1987 ; Robins & Block, 1989 ). Such possibilities stimulate important questions about the diathesis's potential effects on stress. For instance, there are cogent reasons to suspect that diatheses influence the reporting and the generation of life stress.

The third section of the article builds on these insights deduced from examining diathesis—stress models from the perspective of life stress theory and research. We outline several considerations for understanding diathesis—stress interactions. For example, the types of stressors and diatheses, the prevalence of these factors in the general population, and other characteristics of these constructs require elaboration. Instead of general, nonspecific interactive principles, more empirically based hypotheses about diathesis—stress

interactions can be developed for specific disorders. In particular, we use information about the epidemiology of depression and of stress to outline interactive pathways through which people may become depressed. This provides the basis for developing a broader, yet more detailed, framework for understanding diathesis—stress interactions in depression.

After each of these sections we provide recommendations for theory and research specific to the issues raised. We conclude the article with a general discussion of limitations concerning stress and diathesis concepts separately, of models built on these two concepts, and of the implications for theory and research that essays to develop multifactorial models of psychopathology.

## Conceptualization of Life Stress in the Context of a Diathesis

A person's life comprises multifacted circumstances and dynamic processes. The basic task for stress theory is to abstract the characteristics of the concept that lead to disorder from the background turmoil of life's ongoing vicissitudes. Since the creative, but understandably crude, initial efforts at quantifying life stress using life event checklists ( Holmes & Rahe, 1967 ), a great deal of effort has gone into comprehensively specifying the characteristics of life experiences that are believed relevant for precipitating disorder ( Alloy et al., 1988 ; Brown & Harris, 1986 ; B. P. Dohrenwend, Krasnoff, Askenasy, & Dohrenwend, 1978 ; Katschnig, 1986 ; Lazarus & Folkman, 1984 ; Monroe & Roberts, 1990 ; Sarason, Johnson, & Siegel, 1978 ). Much attention also has been devoted to including forms of stress other than major life events, such as chronic stressors ( Brown & Harris, 1978 , 1986 ; Pearlin, 1982 ) or daily hassles ( Kanner, Coyne, Schaefer, & Lazarus, 1981 ; Lazarus & Folkman, 1984 ). Most investigators have availed themselves of such advances in theory and method. For example, a recent extensive discussion of issues involving diathesis—stress research suggests "an adequate test of these theories would require as

*comprehensive* or exhaustive an assessment as possible of potential stressors" ( Alloy et al., 1988 , p. 49).

A comprehensive approach to measuring stress appears both thorough and rigorous. Such approaches, though, may lead to unintended problems as well. Whereas many of life's circumstances may be deemed stressful in one way or another, not all of these uncomfortable conditions necessarily lead to the development of pathology. The central question is whether all forms of life stress are important for activating a diathesis and precipitating disorder. What has not been appreciated is that a comprehensive measurement approach to stress may lead to confusion about the particular diathesis—stress model held by the investigator. Within the current literature, four aspects of life stress are useful for illustrating this potential problem: (a) temporal factors (e.g., acute vs. chronic stressors), (b) dimensional issues (e.g., major vs. minor stressors), (c) qualitative characteristics (e.g., desirable vs. undesirable stressors), and (d) the rules for combining temporal, dimensional, and qualitative aspects of life stress ( Elliot & Eisdorfer, 1982 ).

### Qualities of Life Stress

Not all life experiences lead to disorder. Although the founding fathers of life event research conceptualized increased "readjustment" as the feature responsible for promoting nonspecific vulnerability to virtually any form of illness, recent research indicates that more specific qualities of life experiences are of particular importance for bringing about illness in general, or specific forms of illness ( Brown & Harris, 1986 ; Monroe & Peterman, 1988 ). Not only is the magnitude of events critical, but so may be the particular qualities of events. For example, severe major events that signify loss or exits from one's social field have been found to predict the onset of depression, whereas events that signify danger (but not loss) have been reported to precede episodes of anxiety disorders ( Finlay-Jones & Brown, 1981 ; Paykel, 1982 ).

Even within experiences designated as major and undesirable, then, only a subset are likely to be relevant for vulnerability to a particular form of disorder. The importance of these distinctions becomes clear when examining the manner in which the different components of stress can be united in the operational scheme. For example, cognitive theories of depression single out affiliative and achievement events as being most theoretically relevant ( Abramson et al., 1989 ; Beck, 1987 ; Hammen, Ellicott, Gitlin, & Jaimison, 1989 ; Robins & Block, 1988 ). However, the frequency of major types of these events (i.e., severe interpersonal losses or achievement failures; see Hammen et al., 1989 ) is relatively low compared with the other types of events (e.g., more minor interpersonal or achievement events, or other types of events; Brown & Harris, 1986 ), and even lower compared with the other facets of stress (e.g., chronic stressors or daily hassles). Consequently, if one were to aggregate all forms of stress, or to aggregate major, minor, and chronic forms of interpersonal (or achievement) stress, the importance of the major events would be obscured by the more common components of stress.

The general concern is that as thinking on stress becomes more differentiated and intricate, the implications for diathesi—stress theories must be thought through in consonant ways. In place of generic stress concepts and measures, more specific forms and qualities of the construct should be evaluated. Whereas recent discussions commendably focus on properties of stress that are consonant with specific theory (e.g., Abramson et al., 1989 ), such discussions tend to overlook more general issues involving properties of stress that transcend the particular theory involved. As we illustrate next, specific forms and qualities of stress also have important implications for clarifying how stress might interact with diatheses to produce disorder.

### Temporal Characteristics of Stress

Time is another dimension along which life stressors can be conceptualized. Some experiences are relatively acute, circumscribed events that occur at a

particular point (e.g., deaths, job loss). Others are more chronic or intermittent and defy precise temporal specification (e.g., chronic financial or marital difficulties). Most important, acute and chronic stressors might play different roles in relation to diatheses and the etiology of the disorder.

For acute major events, it is relatively clear why the diathesis is activated and the disorder develops at a particular point (i.e., the event accounts for both the diathesis activation and the timing of disorder onset). For chronic stressors, however, this issue is more problematic: Why, at one particular time, does the diathesis activate and the person succumb? Instead of acute breaking effects of stress, there may be a more gradual, chronic attrition that eventually brings about disorder. One implication of a chronic stress model, then, is that additional components are necessary to account for the timing of breakdown. Another implication parallels the temporal characteristics of the particular disorder under study. For example, chronic forms of adversity may be of particular relevance for chronic forms of disorder (e.g., dysthymia; Brown, Bifulco, Harris, & Bridge, 1986 ), or for disorders with a prolonged developmental course (e.g., coronary heart disease; Monroe, 1989 ; Neilson, Brown, & Marmot, 1989 ).

By virtue of being parsimonious, the acute major event model may be implicit in many views of diathesis—stress interactions. Yet the issue of the type of stress involved remains an empirical question. Most discussions of diathesis—stress theories, though, are simply unclear with respect to the implications of these distinctions between types of stress for disorder onset ( Abramson et al., 1989 ; Alloy et al., 1988 ; Hammen, 1988 ; Robins & Block, 1989 ). Lack of theoretical attention to such differences in the nature of stress leads to imprecision in the assessment of acute and chronic stressors (see below, Perspectives on Perspectives of Stress Measurement section). At least at the present stage of knowledge, these different types of adversity should not be indiscriminately merged.

### Major and Minor Dimensions of Events

Another aspect of life stress currently attracting interest is major versus minor events ( B. P. Dowhrenwend & Shrout, 1985 ; B. S. Dohrenwend, Dohrenwend, Dodson, & Shrout, 1984 ; Kanner et al., 1981 ; Lazarus, 1990 ; Monroe, 1983 ). The latter forms of stress, often referred to as daily hassles, have a high frequency of occurrence as compared with major events and have been found to predict a variety of psychological and physical health outcomes. Some investigators suggest that these forms of stress are better indexes of vulnerability than major life events ( Kanner et al., 1981 ; Lazarus, 1990 ).

Many of the minor events included in existing inventories reflect experiences that also tend to be intermittent or chronic. Although these events can be characterized as low-level intensity, they often represent consistently recurrent problems (e.g., ongoing financial problems) or background chronic conditions (e.g., job dissatisfactions). Again, the issue is raised of how these fit into the operational approach adopted and into the stress model of the process involved. For instance, if hassles are recurrent and chronic (as the test—retest correlations strongly suggest; see Kanner et al., 1981 ; Monroe, 1983 ), the timing of onset is again an issue. Both by definition and recent evidence, daily hassles are a common and recurring component of everyday life. Why at any one point in time might they activate the diathesis as opposed to any other point in time?

In parallel with the previous discussion involving chronic and acute forms of stress, the minor dimensions of life stress appear to be at variance with the implicit major acute event model of stress. Furthermore, there are many challenging considerations involving the measurement of daily hassles, particularly when studied in relation to psychological disturbances (see below; also Brown, 1990 ; B. P. Dohrenwend & Shrout, 1985 ; B. S. Dohrenwend et al., 1984 ; Lazarus, 1990 ; Monroe, 1983 ). Again, merging such stressors with

other types of stress may result in an insensitive or misleading operational scheme.

### Summary: The Conceptualization of Life Stress

The essence of the foregoing discussion is how different facets of life stress should be integrated or differentiated in theory with respect to disorder. In summarizing this issue, we address two major considerations. First is a question of additivity: Do diverse dimensions and qualities of stressful life experiences summate in their impact? Second is a more complex issue pertaining to the association between different forms or qualities of stress over time in the production of disorder: Do different dimensions or qualities of stress interact in ways to increase overall vulnerability?

The issue of additivity traditionally has been restricted to whether disparate types of events are cumulative in their effects or whether any specific subsets of events are significant for disorder. With the inclusion of new dimensions and severities in more recent formulations of stress, this topic may be expanded from totals of major life events to totals of diverse major events, chronic difficulties, and minor stressors. Using the additive approach, people can attain comparable stress scores through exposure to very different psychosocial circumstances. For example, someone experiencing a variety of minor annoyances can be equated in a summary additive operational scheme with a person experiencing one major, devastating experience. Furthermore, in a probabilistic sense, the minor events and chronic stressor–so frequent and prevalent–typically account for the majority of the variance in any person's stress score (as noted before, the major life events occur relatively infrequently; Brown & Harris, 1986 ; Depue & Monroe, 1986 ; Miller et al., 1986 ). Thus, it would seem unwise at the present state of knowledge to equate people reporting many minor difficulties with those reporting but one major life crisis.

The question of interactions between types of stress raises relatively novel questions. It suggests that particular combinations of different stressors may prove especially noxious, above and beyond their independent additive effects. For instance, an increased frequency of minor hassles in the presence of a major life event might produce especially high risk. Alternatively, major life events that "match" severe chronic stressors have been found to be particularly likely to bring about depression (i.e., acute events that arise from or have direct implications for an ongoing difficulty; see Brown & Harris, 1989 ). Interestingly, such possible interactions among stressors indicate ways in which these factors impact over time in the development of disorder. For example, daily hassles in the context of major acute stress may represent one mechanism through which extreme stress impacts on a daily basis (cf. Monroe, 1983 ). Or major events that match a preexisting, ongoing difficulty may be particularly powerful in producing a hopeless cognitive set ( Abramson et al., 1989 ; Brown & Harris, 1986 ). These expanded models of stress effects, too, require evaluation within diathesis—stress formulations.

Ultimately such questions must be anchored within theory for specific disorders. It is unlikely that stress or different components of stress possess uniform consequences across different psychobiologic mechanisms and different forms of disorder ( Depue & Monroe, 1986 ). Thus, different forms of stress may eventually prove to be important for the development of a particular disorder, or for different forms of disorder. This appears particularly true for depression. For example, Brown and Harris (1986) have shown that severe types of events involving loss are highly associated with risk for this disorder. In contrast, these investigators have not found that other types of major life events, or any types of minor life events, contribute to the likelihood of depression. At least for clinical forms of depression, many types of stress may not be etiologically relevant. (This is a point of considerable importance to which we return.) Overall, in light of these studies, one can conclude that conceptual approaches insensitive to distinguishing these different forms and qualities of stress, and

insensitive to the interrelations between these forms of stress in creating vulnerability, will most likely provide diluted and weak tests of diathesis—stress theories.

### Conclusions and Recommendations

Recent conceptualizations of stress proposed for research on the diathesis—stress theories tend to be vague about the nature of stress involved and the types of interactions between stressful circumstances that may be of particular importance in creating vulnerability. Some types of stress represent relatively major events circumscribed in time. Other types of stress represent minor hassles, and still others represent chronic problems. If the stress score is a composite of all of these, it is difficult to pull apart the essential elements. This is especially problematic, in that the majority of items that will be endorsed repeatedly over time are likely to be the chronic and minor events. These facets of stress obscure the types of experiences on which the diathesis—stress theory most parsimoniously rests: the major events or large-scale adverse changes in one's life circumstances.

It could be contended that (a) recent conceptualizations of stress do not preclude a subsequent, more fine-grained analysis or (b) diverse forms of stress many be relevant for depression onset. Although these points have merit, we believe that they miss a central concern. Namely, the conceptualization of stress–its dimensions and qualities–should *guide* the definition of the construct and consequent measurement practices. The nature of the particular stressors, and the role that they play with other forms of stress, requires greater theoretical emphasis in relation to specific diatheses as one enters into the empirical arena. Existing research on life stress has not attended to these distinctions in dimensions of stress. For instance, recent studies and reviews do not distinguish adequately among types of stress at a conceptual level (e.g., Barnett & Gotlib, 1990 ; Holahan & Moos, 1990 ; Neitzl & Harris, 1990 ; Smith, Smoll, & Ptacek, 1990 ), and virtually all such research has not attended to

critical measurement issues for adequately defining and distinguishing between different forms of stress (e.g., Lazarus, 1990 ; Needles & Abramson, 1990 ; Smith et al., 1990 ; see below, Perspectives on Perspectives of Stress Measurement section).

Overall, a comprehensive conceptualization of stress can lead to a vague definition of the construct, which lacks the requisite theoretical specificity for powerful prediction. The propensity to experience the syndrome of depression may have evolved as a response to relatively specific forms of adversity and privation, not as a response to a haphazard assemblage of abrasive and annoying circumstances. Activation of the diathesis, then, may be confined to certain classes of major, biologically meaningful, stimuli or chronic conditions. For other forms of disorder and the respective mechanisms involved, similar specificities of environmental demands are likely to be of importance. Careful attention is required to avoid diluting the stress construct by overinclusion of intuitively appealing, yet theoretically disjointed, aspects of life stress.

## Assessment of Life Stress in the Context of a Diathesis

Without evidence to the contrary, to assume that a diathesis is entirely latent before its activation would be shortsighted. Indeed, a diathesis may influence (a) the measurement of life stress or (b) the generation of life stress. Before addressing these specific concerns, preliminary discussion of assessment and measurement issues in life stress research is required. This lays the groundwork for understanding particular problems concerning diathesis—stress theories when viewed in the context of contemporary life stress research.

### Perspectives on Perspectives on Stress Assessment

A fundamental issue in the assessment of life stress is the reference point for defining what qualifies as stressful. One viewpoint is that the individual (i.e., the subject or respondent) is the person most qualified to define which

circumstances are stressful, and to indicate to what degree they are stressful, in his or her own life. The rationale is that such information is available only to the particular person who experiences the specific life circumstances and that he or she is therefore the best arbiter for such determinations. For convenience and consistency with previous discussions, we term this system of assessment as *respondent based* ( Brown, 1981 ). It is the most common approach currently used in the literature.

The alternative viewpoint is that although the individual has the best access to information pertaining to his or her life, other considerations render the use of the individual's definitions and assessments scientifically problematic. First, the person's perceptions of stress can commonly result from psychopathology. The presence of depression virtually, by definition, ensures that the person perceives stress (because depression goes hand in hand with a view of the world as full of obstacles and unsolvable problems; Beck, 1967 ). Thus, when a person is asked to report on the stress in his or her life, the independent (i.e., stress) and dependent (i.e., depression) variables are too easily fused ( Brown, 1974 ; Monroe & Peterman, 1988 ).

Second, irrespective of potential confounding with depression, respondent-based procedures result in considerable variability in the types of experiences included within ostensibly homogeneous life event categories ( B. P. Dohrenwend, Link, Kern, Shrout, & Markowitz, 1987 ). For example, two people with very different external circumstances could endorse "serious illness in a close family member." For a particularly worrisome person, the response could reflect a child's one-day bout with the flu; for another, the response may reflect a spouse's recent heart attack. Because the perception is the basis for the measurement, the stress score could be identical for the two people, despite dramatically different environmental bases. In fact, the person's interpretation of an item may be so at variance with that intended by the investigator that the person altogether misses the essence of the question (e.g., a woman whose

husband had a heart attack 4 months previously may skip the item, because at the time of the stress assessment he had recovered well and was doing fine and in her view it was therefore not serious). The result is that considerable discrepancies arise between what the respondent labels as an event and what a more objective, or *investigator-based,* view would define as an event ( Brown, 1981 ). In other words, the stress measurement contains considerable variability because of idiosyncratic interpretations by respondents. Recent research bears out the empirical reality and high degree of distortion resulting from this fundamental measurement concern ( B. P. Dohrenwend et al., 1987 ).

This issue is analogous to problems noted previously in regard to classifying depression by self-report methods (see Depue & Monroe, 1978a ). Self-report checklists reflect a limited range of information on the experiences involved and on the biographical circumstances brought to bear in making informed decisions. Subjects reporting on their lives do not have the overall perspective of the investigator in understanding the meaning and evaluating the applicability of the alternative items for endorsement. They thereby bring to the assessment situation a variety of idiosyncratic views that are often at variance with those of the investigator. Diagnostic practices in psychopathology research have taken firm steps toward increased standardization though the development of clear-cut guidelines and operational criteria used by diagnosticians trained in their use. The assessment of life stress should adopt comparable procedures to standardize definitions and to enhance measurement reliability.

Most important for the present discussion, when theory becomes more differentiated with respect to types of stress, these measurement concerns are magnified. Comprehensive approaches to assessing life stress may outstrip the available operational technologies. When different forms of stress are involved, the investigator must ensure reliable differentiation of the respective components. The procedures used to define and operationalize the different forms of stress must be quite explicit about the rules and criteria used to

distinguish between them. For instance, what is deemed major or minor, acute or chronic, clearly cannot be left to the respondent (for the errors noted above may only be compounded) and must be confronted in a direct, elaborate manner. At what point do events that happen more than once become ongoing difficulties (e.g., a serious marital argument every day, week, month, or year)? How are events that happen within the context of an ongoing difficulty distinguished and defined (as opposed to being simply part of the difficulty)? What are the threshold determinants that separate major events, minor events, intermittent events, and chronic difficulties? Extensive rules and explicit guidelines about he time frame involved, frequency of the event, and severity of the circumstances are required to provide an adequate system of measurement. [2]

Overall, this means that the investigator using respondent-based procedures has lost control over the specification of component features of the diathesis—stress model: The stress score represents an uninterpretable blend of the inputs from two correlated but different theoretical domains (i.e., subjective perception and environmental circumstances). In a similar manner, control over specifying the component types of stress is also lost: The stress score represents an unknown blend of different types of stress. Yet, there are two other major reasons why measurement issues require greater attention in diathesis—stress research. These are addressed next.

### The Measurement of Life Stress: Potential Diathetic Biases

Most diathesis—stress theories assume that until stress activates the diathesis, the predisposition is essentially inconsequential. This assumption requires critical scrutiny. For example, the diathesis may influence the person's perception of daily life and thereby his or her reporting of life stress. Such concerns are easily illustrated with a cognitive diathesis (although the general argument is applicable to any form of diathesis). People with a cognitive diathesis by definition view the world through characteristically different

perceptual filters. This point is an extension of the previous one concerning the problems of respondent-based (i.e., subjective) assessments of life stress yet raises a more perilous concern. Individual differences in the reporting of life stress potentially are directly influenced by the diathesis.

There is no assurance, then, that the cognitive diathesis does not systematically influence the perception of stress. This concern applies to three related levels of measurement: (a) What experiences are recalled, (b) what experiences are defined as stressful events, and finally (c) what stress level is associated with the event. Specifically, if one possesses a cognitive vulnerability for certain types of stressors, the person may recall experiences differentially, possess a lower threshold for deeming relatively minor experiences as important life events, and rate such events as possessing greater aversiveness. For example, if a person is cognitively vulnerable to affiliative events, he or she would be more likely to recall such encounters, to perceive minor encounters as more major events, and to subjectively weight such encounters as more aversive. At the extreme, very trivial events, fueled by a markedly potent diathesis, could underlie the relationship to depression.

Portrayed more generally, the situation is one in which two groups are defined on a priori grounds on the basis of the presence or absence of the cognitive diathesis: a vulnerable group and an invulnerable group. Comprehensive measures of life stress are taken and compared. The vulnerable group reports higher stress, but largely because of the elevation of more minor psychosocial conditions to major levels of personal importance (or of inconsequential incidents to daily hassles status). The so-called objective (or investigator-defined) environmental circumstances are comparable for the two groups, but because of the diathetically driven perceptual differences, the respondents' viewpoints of the conditions differ markedly. Note that the differences between the two groups could be amplified further if subjective weights of life events are used for the stress score. Such double-dipping into the influences attributable to

the cognitive predisposition could dramatically inflate the perceived stress scores, without differences in the actual stressful conditions faced by the two groups.

Overall, respondent-based procedures possess serious limitations when investigating life stress within diathesis—stress theories. One cannot ensure that the diathesis does not essentially override the environmental input, to the point that external stress is a minimized, if not meaningless, component of the model. Such confounding may increase the likelihood of a statistical endorsement of the hypothesis (i.e., the operational confounding of the diathesis and the stress score may bias the design toward confirming significant interactions; see Cohen & Wills, 1985 ; Thoits, 1982 ). The measure of stress now represents an unknown amalgam of (a) individual differences in idiosyncratic perceptions (random error), (b) biased perceptions (systematic error), and (c) external environmental circumstances.

### Generation of Life Stress: Potential Diathetic Influences

A diathesis could influence a person's life stress assessment in ways other than simply producing perceptually based differences in self-report data. A very large proportion of life's stressors comprise experiences over which people have some degree of control. Most people are, at least in part, the creators of the circumstances they endure ( Monroe & Peterman, 1988 ; Rutter, 1986 ). The possibility that a diathesis influences the manner in which the person negotiates life's course, and consequently the nature of the stressors to which he or she is exposed, raises penetrating questions for diathesis—stress formulations of depression.

This point is again illustrated most easily with respect to a cognitive diathesis. People have been predicted to be differentially susceptible, depending on their particular cognitive vulnerability to achievement or affiliation events. However, given the hypothesized cognitive vulnerabilities to these types of experiences, it

is quite plausible that the person navigates a life course that promotes differential exposure to the respective areas of vulnerability. For example, someone with a high affiliative vulnerability may be especially sensitized to interpersonal interactions in key relationships. Vigilant to possible signs of impending rejection, he or she makes constant demands for assurance and security. Relatively benign interpersonal exchanges may take on major personal meaning; over time the behavior becomes increasingly cloying, and eventually precipitates the very circumstances it was intended to avoid (i.e., rejection).

In terms of other forms of predisposition, equally important examples place the concept of stress into a more encompassing framework. For instance, people who develop major mood disturbances often display a previous subsyndromal course of affective symptomatology ( Akiskal, Djenderdejian, Rosenthal, & Khani, 1977 ; Depue et al., 1981 ; Klein, Depue, & Slater, 1985 ). The predisposition possesses biologic and familial correlates ( Depue, Kleiman, Davis, Hutchinson, & Krauss, 1985 ; Depue et al., 1981 ; Klein et al., 1985 ). The behavioral manifestations of the predisposition include many of the symptoms of depression and hypomania yet do not meet both the severity and duration criteria to qualify as major episodes of affective disturbance. Nonetheless, these features are sufficiently frequent and severe so as to cause considerable turbulence in the interpersonal and employment spheres of the person's life. For example, irritability, fatigue, lack of concentration, and social withdrawal predispose the person to a myriad of interpersonal and employment problems, potentially resulting in major events such as losses of relationships or of one's occupational livelihood.

Within this example, stress is not a random process, but rather part of a developmental sequence systematically influenced by the diathesis. Whereas the construct of stress may still play an important role in the evolving scheme, it is generated to a considerable degree by the person's behavior, which in turn is

likely to be influenced by the diathesis. Essentially, this places the meaning of the diathesis—stress interactions within a broader interpretive context and requires supplementary information and procedures to understand the implications of these interactions for the development of depression. (We return to this topic below.)

### Conclusions and Recommendations

When stress scores are a composite of an unknown mixture of individual differences in psychosocial circumstances and in perceptual processes, little headway can be made within the diathesis—stress model in understanding the importance of these two contributing components. This problem is exacerbated when distinctive facets of stress also are included in the model. Only when there is some standardization of, or consensually derived agreement on, the definition of stress and the distinctions between components of stress can the implications of individual differences in perception be systematically investigated (see Brown & Harris, 1986 ; B. P. Dohrenwend et al., 1987 ; Miller et al., 1986 ).

A second tier of uncertainty arises from the possibility that the diathesis influences the form or frequency of stress that the individual experiences. This is a more subtle problem that is still essential for understanding the developmental processes through which depression emerges. Principles and procedures are being developed to address this complex concern. For example, a risk group defined according to some diathesis must be compared with controls before the emergence of depression in terms of the frequency and severity of investigator-defined types of events. If differences arise, then the model becomes more complex in depicting the nature of these interactive processes over time. (We take this issue up again, as well as possible solutions, in the final section of the article.) It is clear, though, that this issue has been consistently overlooked and that considerable work remains in understanding the pathways by which diatheses may influence life stress.

Thus far our concern has been with (a) the nature of stress implied by diathesis—stress models and (b) the influence of a diathesis on stress. Theory may be more explicitly developed and more finely adapted for particular forms of stress and may need to be extended to accommodate how the diathesis influences diathesis—stress interactions. As is shown next, when one moves from general models to more specific models for particular disorders, there is information available to refine these ideas further and to elaborate the hypothetical interactive processes.

## Characterizing Diathesis—Stress Interactions

Despite the long-standing appeal of diathesis—stress concepts, the manner in which diatheses and stressors interact to produce disorder remains poorly specified. In the next sections, we first address basic ambiguities in current diathesis—stress formulations. This helps to establish the shortcomings of existing viewpoints and to show concretely how characteristics of stressors and diatheses may be more directly estimated. Next, we draw from existing research on depression to illustrate how better information about diatheses and stressors can be acquired. Finally, we outline the implications of this analysis for developing more detailed models of diathesis—stress interactions for depression.

### Assumptions About Interactions, Diatheses, and Stressors

There are two levels of ambiguity that cloud the issue of interaction in diathesis—stress theories. The first level concerns common assumptions about the nature of the diathesis—stress interaction. The second level is more fundamental, yet subtle. It involves assumptions about the nature of the diatheses and stressors, respectively, that underlie the interaction.

#### Assumptions about interactions.

Although most discussions of diathesis—stress models clearly indicate that both factors are important for producing disorder, the unique importance of their combined impact typically is not described. This leads to considerable ambiguity. For example, it is unclear if such thinking implies (a) simple additivity (i.e., the degree of stress and the loading of the diathesis summate, with virtually any complementary combination of sufficient magnitude producing the disorder), (b) simple interaction (i.e., a synergism between the diathesis and stress that yields an effect beyond their combined separate effects; Rothman, 1976 ); or (c) complex interaction (i.e., various combinations of additivity and synergism, such as postulating threshold effects for the diathesis).

This verbal vagueness can be clarified algebraically. Assuming that the influence of stress (ST) is at least partly conditional on the diathesis (DS) in relation to depression (DEP): [3]

This general model is illustrated in Figure 1 . The effects of stress are dependent on the diathetic loading. Typical discussions of life stress and diatheses, however, often imply only the additive portion of this model (i.e., $b_3 = 0$ ). The degree of diathetic loading can be offset or compensated by the degree of stress (and vice versa). A simplified case, wherein the diathesis is portrayed as dichotomous (i.e., high or low), is illustrated in Figure 2 . People with an extreme diathetic loading (Individuals x) require only minimal stress ( $b_x$ ) for activation and a high probability of disorder onset ( $a$ ). People with low diathetic loading (Individuals $y$ ) require more extreme levels of stress ( $b_y$ ) for activation of the diathesis and a comparable probability of depression onset ( $a$ ). This interpretation fails to distinguish between conditions under which the joint effects of the two factors are additive versus synergistic (i.e., the joint effect exceeds the sum of the separate effects; Rothman, 1976 ). It does not use the diathesis—stress concept. This "titration" interpretation of the model is compatible with earliest formulations of "nervous diseases" (e.g., Whytt, 1765 ), which was more recently promoted by Slater and Slater (1944) , and is

congruent with many contemporary descriptions of diathesis—stress theory ( Abramson et al., 1989 ; Davison & Neal, 1990 ; Zubin & Spring, 1977 ) and research ( Pollitt, 1972 ; Stenstedt, 1952 ).

Even when the theory embodies true interaction (i.e., $b_3 \neq 0$ ), differing assumptions about the diatheses and stressors influence the hypothesized form of their interaction, which in turn leads to added ambiguity for the type of model portrayed.

**Assumptions about diatheses and stressors.**

Probably as a result of early conceptualizations of the physiology of stress ( Selye, 1936 ) and of life event stress ( Holmes & Rahe, 1967 ), stress typically has been viewed as nonspecific, varying only in degree (from low through high levels), and a very common aspect of everyday life. This nonspecific conceptualization of the construct is consonant with most early and many recent theoretical formulations (e.g., Davison & Neale, 1990 ; Lazarus & Folkman, 1984 ; Slater & Slater, 1944 ; Zubin & Spring, 1977 ). Given such assumptions, stress would not be a very discriminating factor in the diathesis—stress model (see Guze, 1989 ; Heston, 1988 ).

In contrast, the diathesis is often assumed to possess very different characteristics. First, some viewpoints hold that a diathetic threshold exists: People who fall below the threshold will not develop the disorder, whereas those above the threshold are vulnerable ( Bebbington, 1987 ). Second, the prevalence of above-threshold diathetic loading is inferred to be relatively restricted in the general population (i.e., it is believed to be present among a minority of people). Overall, this characterization, too, is consonant with many discussions of diathesis—stress premises ( Meehl, 1962 ; Slater & Slater, 1944 ; Zubin & Spring, 1977 ), with genetic models of liability ( Bebbington, 1987 ; Falconer, 1965 ; Slater & Cowie, 1971 ), and with many if not most current views on biological diatheses for major psychiatric disorders (e.g., Guze, 1989 ;

Heston, 1988 ; Reich, Cloninger, & Guze, 1975 ; Whybrow, Akiskal, & McKinney, 1984 ). [4]

If stress is continuous and the diathesis is discontinuous, then more specific models of interaction are indicated. The diathesis is a necessary, but alone insufficient, component of the scheme. Stress is relevant only once above-threshold value of the diathesis is met. This portrayal of the diathesis—stress interaction suggests at least two additional models. [5]

In the least complicated case, the diathesis operates in an all-or-none manner: present or absent. The values for $b_2$ in Equation 1 become either 0 (diathesis absent) or 1 (diathesis present). This permits Equation 1 to be rewritten in terms of these assumed values of the diathesis (DS):

The threshold effect, however, specifies that stress is only operative conditional on the diathesis. When the diathesis is absent, there is no main effect for stress. Thus, when the diathesis is absent (DS = 0), $b_1$ in Equation 2 is constrained to equal 0. Furthermore, given a pure interaction model (stress and diathesis are only relevant when both are present), there are no main effects (even when DS = 1, $b_1 = b_2 = 0$ ). Equation 2 then becomes the following:

This is portrayed in Figure 3 . Without the diathesis, disorder is not possible; with the diathesis, expression of disorder is conditional on degree of stress. Viewing the interaction in this manner is useful for raising a question concerning the assumed representation of the diathesis: What proportion of the population is represented by the DS = 1 (diathesis present) condition?

The second possibility that follows from assuming a threshold is more general. Instead of being dichotomous, the diathesis is "quasi-continuous." There is a threshold effect, but there is also a continuous effect of the diathesis once the threshold is superseded. In terms of Equation 1 , this means that $b_2 = 0$ (diathesis absent) or $b_2 \neq 0$ (diathesis present). Rewriting Equation 1 :

Once again, imposing the constraint following from the threshold effect that if DS = 0, $b_1 = 0$ and assuming pure interaction $b_1 = b_2 = 0$ :

Finally, because the diathesis is continuous once the threshold is exceeded, the diathesis takes on an infinite number of possible values. Consequently, there is an infinite number of equations for each value of DS $\neq 0$. Rewriting Equation 5 to parallel Equation 3 and allowing DS = $i$ ( $i$ representing the range of values for DS when DS $\neq 0$):

A family of regression lines describes the relationships between the diathesis and stress for different levels of the diathesis. This is represented schematically in Figure 4 . We can now expand on the question raised above with respect to the dichotomous model about the proportion of the population with the diathesis (DS = 1) to include the frequency of people possessing differing values of potency for the diathesis (DS = $i$ ).

Overall, the diathesis—stress model comprises a family of possibilities that portray different relationships between diatheses and stressors. By clarifying aspects of the proposed diatheses and stressors, we confront important issues about their characteristics in the population. First is the question of a threshold and its distribution (i.e., who is vulnerable, who is not?). Second is the question of the distribution of above-threshold loadings (i.e., who is highly vulnerable, who is not?). Interpretations of interactions between diatheses and stressors, though, differ depending on the particular disorder under study. For example, the diathesis and stress for schizophrenia are almost undoubtedly different from those for depression. The nature of the diathesis in the former case is probably more restricted in the general population (and thereby may represent the "rate-limiting" element in the predictive model). In the latter case for depression, the prevalence of the diathesis may be more common (and thereby less of a primary determinant of who develops the disorder). For specific disorders, then, the characteristics of the particular stressors and diatheses involved suggest

how the interaction can be more precisely conceptualized and systematically investigated.

### Parameters of Predisposition to Depression: Diatheses and Stressors

We may now ask whether existing diathesis—stress theories fit with the available information specifically for depression. Current evidence suggests not. Knowledge about the incidence and prevalence of depression, as well as about the nature and prevalence of the stressors, and diatheses found to predispose to this disorder, delimits and more precisely specifies the range of alternative interpretations of diathesis—stress interactions. The main purpose of this section is to provide preliminary form to these ideas. First, with respect to depression, sufficient information exists for developing more specific models. Limited as current data may be, they provide an empirical foundation for assumptions about diatheses, stressors, and their interactions. Second, we again take up the concern that the diathesis may influence the nature or degree of stress to which the person is exposed. This possibility enlarges the interpretative scope for understanding diathesis—stress interactions. Essentially, another layer of issues arises for developing working hypotheses, research strategies, and prevention programs that are based on the nature of the multiple interactions involved. It is of interest, then, to profile implications that follow from each alternative position (i.e., stress and diatheses are dependent or independent).

Before proceeding, we should emphasize that clinical depression is commonly considered to comprise an etiologically heterogeneous group of disturbances sharing common phenotypic characteristics ( Depue & Monroe, 1978a , 1978b ). Any discussion of the base rate of disorder (or diatheses) should be tempered by an awareness that subgroup differences may alter the more general picture. For example, the estimates for the prevalence of depression and their implications for estimates of diatheses that we discuss next may be applicable to only a subgroup of people currently classified as depressed. This would then

modify the particulars of our argument, whereas the logic of the analysis would remain applicable to the designated subtypes. Despite over 60 years of published debate on the topic, though, little consensus has been reached on the debate between the unitary and nonunitary views of nonpsychotic unipolar depression ( Farmer & McGuffin, 1989 ; Kendell, 1976 ). Given such controversy and lack of consistent agreement on proposed subtypes, it seems prudent at present to develop the framework on the basis of the general condition. From this initial reference point, modifications may be made more readily in accord with research documenting the validity of particular subtype distinctions.

**Characterizing the diathesis for depression.**

A major question about the diathesis for depression concerns its prevalence in the general population. Is the susceptibility to becoming clinically depressed relatively confined to an unfortunate few with strong diathetic loading, or is it more widely distributed as part of the psychobiology of being human ( Monroe, 1990 )? Unfortunately, the diatheses proposed for depression are insufficiently specified and operationalized to confidently infer their distributions within the general population. However, lower bound estimates of their prevalence can be tied to estimates of that to which the person is predisposed: depression.

One common viewpoint in the psychiatric literature is that the diathesis for depression is relatively restricted in the general population (see Cooke, 1987 ; Guze, 1989 ; Heston, 1988 ). By virtue of its selectivity, it is the pivotal component (i.e., necessary and prevalence limiting) in the arrangement of etiologic factors leading to an episode of depression. Life stress is viewed at most as a mere precipitant of the disorder and is thereby conceived as only a comparatively trivial element in the etiologic process. There are several sources of indirect support for this point of view. One of the most consistent findings in depression research is the tendency of the disorder to run in families and for the morbidity risk of depression to be higher in such families as compared with the

general population ( Andreasen, 1987 ; McGuffin & Katz, 1989 ). Also, many studies have noted that despite high levels of stress, only a certain percentage of people succumb to depression ( Brown & Harris, 1978 , 1986 ; Paykel, 1982 ). Finally, strong evidence exists for the genetic transmission for at least some forms of bipolar disorder, which is most commonly manifested as the unipolar phenotype within the family pedigree ( Baron et al., 1987 ; Depue & Monroe, 1978b ). On the basis of these sources of information, one might infer that for depression (a) the diathesis is strongly associated with genetic and familial transmission, (b) the diathesis is likely to be relatively restricted in the general population, and (c) stress leads to depression primarily within the population of the predisposed ( Clayton, 1986 ; Guze, 1989 ; Heston, 1988 ; Whybrow et al., 1984 ).

In contrast, other sources of evidence suggest that a diathesis for depression is a more common characteristic of the human constitution and condition. With respect to incidence and prevalence of the disorder, virtually all epidemiologic studies cite high rates in relation to other psychiatric disorders in the general population (e.g., Boyd & Weissman, 1981 ; Charney & Weissman, 1988 ). Furthermore, an unknown proportion of the predisposed never manifest the disorder. For estimating the prevalence of predisposition to depression, the indexes that are based on lifetime risk data provide a reasonable, lower bound estimate. Worldwide, the lifetime-expectancy estimates for major depression range from 8% to 12% for males and from 20% to 25% for females ( Charney & Weissman, 1988 ). Additionally, recent research has documented secular trends in the rise of depression ( Gershon, Hamovit, Guroff, & Nurnberger, 1986 ; Hagnell, Lanke, Rorsman, & Öjesjö, 1982 ; Klerman, 1988 ). In particular, for people born since World War II, depression is more common and occurs at an earlier age. These changes in the epidemiology of depression cannot be explained by simple genetic theories of etiology and a shift in the genetic diathesis (because the time periods involved clearly are not sufficient to account for necessary changes in the gene pool). Instead, some form of gene—

environment interaction has been hypothesized, with unknown environmental factors shifting to account for recent increases in depression in general ( Klerman, 1988 ) or in the incidence of particular subtypes of the disorder ( Giles et al., 1989 ). Overall, then (a) the high frequency of depression, (b) the increasing incidence of the disorder, and (c) the fact that all calculations necessarily underestimate the pool of predisposed people argue for a substantial proportion of people who possess a diathesis for depression.

Note that this line of reasoning is based on a nonspecific view of the diathesis. If different diatheses exist, qualifications in the argument are introduced. For example, biologic and cognitive diatheses may operate by means of separate mechanisms. The high frequency of depression would then be due to the combined prevalence of the two (or more) forms of diathesis, and either predisposing element alone would be represented in the population at a proportionately decreased frequency. [6] (This concern parallels in several respects that noted previously with respect to the issue of etiologic heterogeneity.) Unlike the situation for life stress, though, there is little firm information yet available on which to elaborate the implications of this point. For example, although cognitive and biological diatheses have been proposed, there is little basis for discerning if they represent separate vulnerabilities or different levels of the same vulnerability The existence of multiple diatheses, though, would not negate the general framework we discuss for studying specific diathesis—stress interactions; it would simply require differentiating the framework in accord with the characteristics of the different diatheses. Because there is no firm evidence yet to distinguish between multiple diatheses, the present analysis is based conservatively on the more general condition consonant with the existing literature.

Overall, depression–and consequently the predisposition to depression–is not restricted to a small segment of the population. This is to some extent in contrast to the viewpoint that the rate-limiting factor of the diathesis—stress

interaction is predominately the diathesis ( Akiskal, 1988 ; Clayton, 1986 ; Guze, 1989 ; Klerman, 1983 ; Whybrow et al., 1984 ). Although there may be subgroups of people with especially potent diatheses for depression in general or for subtypes of depression in particular, they cannot fully account for the widespread incidence of the disorder. A substantial proportion of people must possess a diathesis for depression. [7]

**Characterizing the stressors for depression.**

Concepts of stress have most often been presented as nonspecific in form and as a continuous construct comprising a myriad of experiences (see earlier section, Conceptualization of Life Stress in the Context of a Diathesis). However, regarding depression, this is at variance with the available empirical evidence. More specific qualities and dimensions of stress tend to account for the association with clinical depression ( Brown & Harris, 1978 , 1986 ; Cooke & Hole, 1983 ; Finlay-Joness, 1981 ; Lloyd, 1980 ; Monroe, 1990 ; Paykel, 1982 ). In particular, severely threatening events–especially those that involve major loss of valued persons or roles—represent a class of circumstances that appear to be especially effective for eliciting the psychobiologic response (see Brown & Harris, 1986 ; Cooke & Hole, 1983 ; Finlay-Joness, 1981 ; Paykel, 1982 ). The most extensive series of inquiries on this topic has been conducted by Brown and Harris over the past 20 years. They have used sophisticated methods for assessing life stress, have conducted investigations across different samples and cultures, and have interpreted the findings from replications by their research unit and other investigators using the same methods. They conclude that "it is the impact of just one event or difficulty of a sufficient severity that appears to be critical" ( Brown & Harris, 1986 , p. 138). Less severe forms of stress, or hassles and low-level chronic problems, are not important (cf. Bebbington et al., 1988 ).

These findings suggest a threshold for severity and specificity of socioenvironmental circumstances that precede depression. Qualitatively

distinct experiences, in terms of severity and nature, appear to be essential. Lower levels of stress were not found to summate to produce depression. Estimates of the incidence of such forms of life stress therefore can be made. For a 38-week period preceding disorder onset or interview, Brown and Harris (1978) found that severe events or severe difficulties occurred for 75% of psychiatric patients, 89% of onset cases in the community, and 30% of normal or subsyndromal women in the community. These findings are essentially replicated in 9 subsequent studies performed by a range of investigators using Life Events and Difficulties Schedule (LEDS) methodology. Across these studies, one or more severe events or major difficulties occurring before onset ranged for cases from 62% to 94% (average 82%) and for noncases from 25% to 39% (average 33%; Brown & Harris, 1989 , p. 55). [8] Eleven studies of psychiatric disorder using other life stress assessment methods yet including only measures of events (i.e., no difficulties) yielded comparable, albeit less dramatic, results (e.g., and average of 54% of cases experienced major stressful events before onset as compared with an average of 18% of noncases before interview; Brown & Harris, 1989 , p. 56). [9]

Overall, people in the general population are exposed relatively infrequently, not constantly or inevitably, to the life stressors capable of eliciting depression. In other words (a) relatively specific forms of life stress appear to precede depression, (b) there is a threshold of severity for these events, and (c) these types of events are not ubiquitous in the general population. This is in direct contrast to one common conceptualization of life stress (cf. Guze, 1989 ; Heston, 1988 ) and, paradoxically, is more akin to the manner in which the diathesis has been portrayed.

### Synthesis and Speculation

For depression, life stress is characterized as operating in a relatively qualitative manner with a severity threshold. But we do not yet know if further above-threshold gradations are important and, if so, how common such

influences might be. With regard to diatheses, a relatively large proportion of the general population possesses the psychobiologic substrate for developing depression. But, again, we do not know if there is a threshold; if gradations in loading of the diathesis are relevant; and if they are relevant, the prevalence of the differing levels in the general population. We may now return to existing evidence to synthesize speculative conclusions regarding gradations in strength of these factors and to derive implications for refining ideas about stressors and diathesis in depression.

As indicated previously, most depressions follow from one severe event ( Brown & Harris, 1986 ). From the perspective of the population of depressed persons, this indeed appears to be true. For example, Table 1 illustrates that the majority of depressed people in the Camberwell study of Brown and Harris (1978) experienced but one severe event before onset of their depression. [10] Relatively few women experienced more than one event. Therefore, the frequency of depressed women with zero, one, two, or more severe events decreases considerably and proportionately.

From the perspective of individual depressed persons, however, an important complementary interpretation can be drawn. Table 2 represents the same data, rearranged according to the frequency of women depressed (or not depressed) having experienced zero, one, two, or more severe events. The potency of the effects of life stress clearly increases proportionately with the higher levels of exposure. Indeed, of the 7 people who experienced more than three severe life events, all subsequently became depressed (see Table 2 ). Thus, whereas the majority of people who become depressed in response to life stress do so after one major severe event ( Table 1 ), the minority of people exposed to multiple life stressors are at even greater risk in proportion to the number of severe events encountered ( Table 2 ). This suggests a quasi-continuous effect for above-threshold values of life stress.

Although these particular findings require replication, two tentative but very important implications are noteworthy. First and most apparent, there may be above-threshold gradations in the severity of stress that contribute to the development of depression. Whereas a qualitative and quantitative threshold appears to exist, the effect is not purely dichotomous. Multiple severe stressors, although occurring relatively infrequently in a normative sense, may be disproportionately virulent for precipitating an episode of depression. The second point is more speculative and tentative but theoretically provocative. Under increasingly severe levels of these stressors, most if not all people break down. This again implies that the capacity to become clinically depressed is widely distributed in the general population. Although there may be important reasons to moderate this conclusion (to be addressed shortly), the major thrust of the argument remains the same. The ability to become clinically depressed appears quite common and not solely the province of an unfortunate few who possess a particularly pernicious form of the diathesis. [11]

Overall, research-based characterizations of the diatheses and stressors for depression are in marked contrast to prevailing viewpoints. The widespread incidence of depression, the greater prevalence of the predisposition to the disorder, and the frequency and potency of the forms of stress found to precede the disorder all indicate that more specific and empirically based models of diathesis—stress interactions can be developed to guide future research. We next begin to outline such a framework for inquiry.

### Characteristics of Diathesis—Stress Interactions in Depression

If the type of stress required to initiate an episode is not rare yet also not common and if the diathesis for depression is relatively common, then the characteristics of diathesis—stress interactions in the population take on different forms. On the basis of the reasoning just outlined, we assume for heuristic and descriptive purposes that the form of stress initiating depression is relatively uncommon and the diathesis for depression is relatively common. Yet

one issue complicates the interpretive matrix. If the diathesis influences the stress to which a person is exposed, the interactions must be evaluated within a broader conceptual framework that encompasses the developmental dynamics between the diathesis and stress ( Koopman, 1977 ). Because the influence of the diathesis on stress is currently based only on conjecture, we outline interpretive premises and illustrate implications that are predicated on both independence and dependence assumptions.

**Independence of the diathesis and stress.**

The more common the predispositional factor is in the population, the less it explains the distribution of cases ( Rose, 1985 ). This is because the more frequently a causal agent occurs, the less it is capable of explaining the allocation of the particular disorder in that population. Under such conditions, greater explanatory potential resides within the elements of the model that are distributed in a more differentiating manner. For understanding depression, under the presently specified diathesis—stress conditions–i.e., the requisite stress is relatively uncommon, and diathesis is relatively common–life stress may be the more "prevalence limiting."

This characterization of the diathesis and stress, though, raises a somewhat counterintuitive point. To the extent that most people possess the diathesis, the majority of people who become depressed in the general population may be people who possess lower levels of the diathesis. When the diathesis is common and when the activating stress is independently distributed across people (i.e., stress is not influenced by the diathesis), the sheer number of people at lower levels of diathetic risk experiencing significant stressors may offset those at higher levels of risk experiencing significant stressors. Many people at small risk produce more cases of the disorder than few people at high risk ( Rose, 1985 ). [12]

The high-risk paradigm is frequently adopted in this area of research, wherein a predispositional factor is chosen and people are selected for study on the basis of a high loading of the diathetic risk variable (e.g., Alloy et al., 1988 ; Buchsbaum, Coursey, & Murphy, 1976 ). People with the outermost scores on the diathetic variable, however, may not be representative of the majority of people predisposed to the disorder or of the preponderance of people who eventually develop the disorder. Although the processes that give rise to depression in the highly predisposed may be informative theoretically for the conditions under which the less predisposed succumb, there also may be important differences. For example, the constellation of etiologic forces that are sufficient to precipitate depression in the high-diathesis risk group could be different from those required for the lower diathesis risk people.

The practical implications of such a possibility also merit consideration. For example, from a public health perspective, those who give rise to the majority of cases (i.e., the less predisposed by the diathesis) constitute the natural focus of attention for intervention. Irrespective of theoretical commonalities between groups at very different levels of the diathetic factor, the differing combination of diathetic factors between people still suggests potentially important differences in conceptualizing intervention efforts. For instance, because the incidence of relatively specific types of stress within the current framework operates as the more pivotal factor, targeting reduction in its incidence or the manner in which it is diminished or resolved could be more effective prevention strategy than attempting to alter the diathesis (cf. Guze, 1989 ). Again, we caution that such ideas are based on our previously derived estimates and await further clarification. These points serve to provide a very different, yet quite plausible, perspective for diathesis—stress models, given current information and theory.

Assuming independence of the diathesis and stress, though, may oversimplify diathesis—stress interactions. When the diathesis influences stress, the matrix

of interpretational issues enlarges considerably, and by necessity so do the conceptual and research demands required.

**Dependence of the diathesis and stress.**

When the diathesis influences the relevant stressors, diathesis—stress associations become more complex. One general conclusion is that in contrast to the case in which the diathesis and stress are independent, the likelihood of incurring the stressor increases with the loading of the diathesis. To the extent that the diathesis influences the incidence of the requisite forms of stress, the more likely it is that highly predisposed people will develop depression. Under these conditions, proportionately more people who become depressed in the general population are people who possess the highest risk in terms of the diathesis. [13] This point has led to divergent interpretations of existing research and to major misunderstandings between investigators of different theoretical inclinations concerning the relative importance of stress and diatheses in the etiology of depression.

There are at least three alternative viewpoints of the temporal interplay between stress and the diathesis. First, in keeping with the spirit of the diathesis—stress principles, both the diathesis and the stress together constitute a necessary condition for depression onset (see Figure 5 ). Neither alone is sufficient. Although the stress in part follows from the behavioral concomitants of the diathesis, the stress still features as an integral element in the developmental scheme. For example, the case portrayed previously for an affiliative diathesis and the life event of rejection illustrates the issue well: A person who has the cognitive vulnerability to interpersonal events may conduct such activities in ways that increase the likelihood of severe events occurring. The person's attempts to avoid rejection, for instance, through continual requests for reassurance and support, promote the eventual rejection. Both factors together–the affiliative diathesis and the particular stressor–are sufficient. Within this scheme, the diathesis operates at two levels in the model. It enhances the

likelihood of the very conditions occurring that are also necessary to transform it into depression (pathway *a* ). Note, however, that diathesis-driven events are not necessary to produce depression, because events that happen irrespective of the diathesis or person's actions may also serve to activate the diathesis and promote depression (pathway *b* ).

A second viewpoint is that the only necessary factor is the diathesis ( Figure 6 ). Stress is either a minor factor, a result of the diathesis's expression (pathway *a* ), or simply a consequence of the emerging depression (pathway *b* or *c* ). Indeed, this is the radical cognitive view summarized by Epictetus in *The Enchiridion:* "Men are not disturbed by things but by the views which they take of them." Similarly, some biologic models and classification schemes view stressors as irrelevant to the development of at least certain subtypes of depression and define subtypes on the basis of the absence of external stress (e.g., endogenous depression; Carney, Roth, & Garside, 1965 ). Within these perspectives, stressors are only minor contributors to etiology or are essentially epiphenomenal. A predisposed person may become irritable and socially withdrawn and experience concentration difficulties, crying spells, and sleep disturbance. Although primary relationships may be strained and arguments, fights, or even breakups occur, the depression had already begun its insidious course. The stressors, although distressing, were a consequence of the diathesis-influenced prodrome (pathway *b* ) or the onslaught of the disorder (pathway *c* ) and therefore are not viewed as an important element in determining the depression's emergence (e.g., Akiskal, 1988 ; Guze, 1989 ).

The third position posits that the only necessary factor for depression is life stress ( Figure 7 ). Specific stressors are seen as essential to the onset of depression. The diathesis is nonessential, or less important, in the theoretical scheme. Depression can occur irrespective of the diathetic loading (pathway *b* ). What is viewed as the diathesis, however, might be reconceptualized as stress propensity. Attributes of the person (or of his or her social context) give

rise to processes that increase the likelihood of the requisite forms of stress occurring (pathway *a* ). The activation of the diathesis is essentially irrelevant compared with the generative potential of the diathesis for contributing to the circumstances that would give rise to depression (irrespective of the diathesis). The cognitive or biologic propensities previously viewed as diatheses are operative in the model only in so far as they contribute to the incidence of the forms of stress required for depression. Although theoretically unnecessary, the generative potential associated with the diathesis may be an important mechanism by means of which the necessary conditions for producing depression are fulfilled.

Each of these three depictions of diathesis—stress associations yields a variety of theoretical and practical implications. One common point across the different interpretations exists. To the degree that the diathesis influences stress, the high-risk paradigm moves toward appropriately targeting the majority of people who will in all likelihood develop the disorder. This means that there is a tighter correspondence between the people most often selected for study with such procedures and the people most likely to become depressed. It also suggests public-health-relevant approaches for targeting the most important groups for prevention purposes. What is required is a research agenda directed toward greater elucidation of the types and degrees of association between the proposed diatheses and stressors. Such approaches require prospective longitudinal designs, wherein the diatheses are assessed before stress occurrences and the implications of the different models outlined can be tested.

## Conclusions and Recommendations

We have outlined in a broad manner the developmental pathways between diatheses and stressors in depression that fit with current empirical information. But the particular concern for diathetic influences on stress has few data at present on which to base opinions, let alone conclusions. Indeed, methodologies for studying such influences are only beginning to emerge. This

means that in concert with the development of concepts and basic stress measures, additional thought and assessment should be devoted to the specific task of understanding the nature of the temporal interplay between hypothesized diatheses and forms of stress. Whereas much of this latter topic will be at least in part a function of the particular diathesis of interest, the more general issue pertains to the question of how life's stressors begin in the first place.

At one level or another, most life events and chronic difficulties can be traced to some aspect of the person's behavior. As indicated previously, to varying degrees most people are in part creators and perpetrators of the circumstances they endure ( Monroe & Peterman, 1988 ). This is not to say that all stress is merely a by-product of the person's mistakes, failings, or diatheses. But stressors, behaviors, and social environments can work together in complex and internecine ways to initiate, and perpetuate, adversities. At a very gross level, distinctions can be made relatively easily. For example, the death of a close friend because of a natural disaster would not likely be immediately related to the subject's behavior or hypothesized diathesis. But at other levels applicable to more common life events, such judgments become more difficult. For example, a person may be laid off because of an industry-wide initiative. However, only 70% of the employees may actually be dismissed, perhaps selectively on the basis of past performance. How much of the event is attributable to the person? In a similar vein, relationship difficulties and breakups, which often reflect processes of many years' duration, are very hard to ascribe cleanly to one person or another, one shortcoming or another. A very large part of life's misfortunes are end points of such multidetermined processes.

Procedures for handling these concerns have begun to develop. For example, investigators have devised rating systems to describe the degree to which an event might result from a preexisting disorder, or the behavior, of the person (

Brown & Birley, 1968 ; Brown & Harris, 1986 , 1989 ; Miller et al., 1986 ). These systems are useful for eliminating major confounds (e.g., events that are an obvious consequence of depression), but as yet they typically are not adequate to address the full array of complicated possibilities for interplay between diatheses and stress over prolonged periods of time. Events that result from the combination of many factors, only one of which may be the person's behavior, possess an unclear status. Are they epiphenomenal by-products of the diathesis, or are they integral components of the developmental process? More differentiated systems are required to study the influential forces in the origins of life events and to use such information to better understand the potential interactive pathways between diatheses, stress, and psychopathology (see Brown & Harris, 1989 ).

Recent work explicitly addressing diathesis—stress premises strongly affirms the need for such efforts ( Bebbington, et al., 1988 ; McGuffin, Katz, Aldrich, & Bebbington, 1988 ; McGuffin, Katz, & Bebbington, 1988 ). Using the LEDS life event assessment system, McGuffin, Katz, and Bebbington studied the relationship between life events and depression in 83 families identified through depressed probands. In contrast to a traditional diathesis—stress prediction, they found no support for an inverse relationship between the presence of family loading for depression (the diathesis) and life adversities preceding the subject's depression (the stress). In fact, "the relatives of probands whose onset of depression followed life events or chronic difficulties had slightly higher lifetime rates of depression than the relatives of probands whose onset was not associated with adversity" ( McGuffin, Katz, & Bebbington, 1988 ; p. 775). They also found relatives of depressed probands to have significantly raised rates of both current depression and recent threatening life stress. In other words, both the liability to depression and the propensity to experience severe life stress apparently were familial. [14] More refined procedures for determining the origins of events, from the personal influence through that of the broader social

structures, and the nature of their interactive impact with the diathesis are required.

## Concluding Remarks

Over 25 years ago, David Rosenthal (1963) described diathesis—stress theories as "the ones in which genuine meaning attaches to the commonly repeated statement that heredity and environment interact" (p. 509). These theories are heuristic devices for developing an understanding of how predispositional factors from different domains heighten susceptibility and, eventually, create sufficient conditions for disorder onset. This represents the promise of such ideas. It is this interactive aspect, we believe, that has captured the minds of investigators and represents the guiding principle of the premise. However, with respect to these theories, Rosenthal (1963) further noted that "the great majority of them are also exasperatingly loose, since the nature of the predispositions and the stressors, as well as the mechanisms of interaction, are usually only vaguely conceived or formulated" (p. 509). It is this disintegrative accompaniment to such thinking that has impeded progress.

The looseness associated with these theories may be addressed in a direct manner. As illustrated throughout the article, the diatheses and stressors can be better conceptualized and more precisely measured with respect to specific forms of psychopathology. For example, the generic notion of stress can give way to particular types of stressors that are most relevant for achieving an understanding of the disorder of depression. In a complementary vein, further specification of specific types of diatheses will also contribute to the development of more precise and useful models. Better determinations of the diathesis through family history ( McGuffin, Katz, & Bebbington, 1988 ), biological markers ( Monroe & Depue, 1991 ), individual differences in cognitive factors ( Abramson et al., 1989 ; Beck, 1987 ), or other domains of social and psychological vulnerability ( Brown & Harris, 1989 ) will help to clarify these

issues. Finally, through focusing on specific qualities of stress in relation to the specific disorder of depression, we profiled alternative models for depicting diathesis—stress interactions. These possibilities make more explicit the pathways that future research should address to develop a better understanding of the meaning and importance of such interactions. In turn, research findings that are based on such approaches will contribute to more informed programs for risk detection and prevention. These interactive issues bring the focus of the diathesis—stress premise back to its conceptual essence: the nature of the interaction between elements in the etiologic process over time.

There are more subtle and pervasive problems, though, that also give rise to the "looseness" that accompanies diathesis—stress theories. These concerns are not so easily eliminated through the direct methods we have advocated. Rather, they are much broader and diffuse, involving the permissive conceptual structure that gives rise to causal explanations in psychopathology research. The concept of life stress can be sufficiently amorphous and can be sufficiently imprecisely defined and measured so as to fit into virtually any explanatory scheme. We have shown that systematic errors (e.g., confounding with diatheses or disorder) and unsystematic errors (e.g., composite measurement procedures) can easily yield mixed and confusing findings. The idea of a diathesis, in the abstract and in the applied, suffers from similar susceptibilities of imprecision in concept and measure. Together, stress (a collective representation of exogenous considerations) and diatheses (a collective representation of endogenous considerations) essentially cover the vistas of explanatory concepts in contemporary psychopathology research. Left only partially constrained in theory and measure, they can unite as powerful co-conspirators in nonexplanation.

Present-day research on depression has expanded the list of contributory considerations from the two-factor diathesis—stress model to more

encompassing multifactorial representations ( Akiskal, 1985 ; Akiskal & McKinney, 1975 ; Depue, 1979 ). The factors found to be associated with depression are quite extensive, involving aspects of early developmental experiences, various biologic vulnerabilities, diverse psychological susceptibilities, and other types of socioenvironmental contributions. These ideas no doubt touch on additional elements of potential import and can assist in developing a broader understanding of the disorders. Yet multifactorial models simultaneously inflate the problem inherent in the relatively simple two-factor model just reviewed. If one of these factors, one pairing of these factors, or one complex of these factors is not found to predict disorder, then another factor, pairing of factors, or complex of factors is readily raised (see Meehl, 1977 ). Ultimately, within such a broad explanatory system there can exist an expanding array of interactive and additive alternatives to account for virtually any arrangement of findings.

Finally, the potential for ambiguity resides not only in the realm of these independent variables and their interactions (i.e., stress, diatheses). Most conceptualizations of the dependent variable, disorder, cautiously acknowledge that multiple subtypes are likely to exist ( Weiner, 1977 ). Again, although this probably represents a reality of such conditions, it also injects another layer of potential looseness. The problem of interchangeable elements within the independent variable is mirrored by interchangeable categories of the dependent variable. If the findings are not in theoretical accord for any particular outcome, different subtypes of the disorder are invoked, which in turn can be explained by a different factor, pair of factors, multitude of factors, and so on. A symmetry for misunderstanding exists between the independent and dependent variables so that hypotheses are simultaneously elusive (difficult to prove) and evasive (difficult to disprove).

Of course, if the component concepts of the multifactorial model are reasonably well understood, if their operationalizations represent reasonable

approximations, and if the subtypes of the disorder are amply outlined, concerns such as these are far less compelling. Various hypotheses could be refuted because investigators would have an acceptable hold, both in theory and in measure, on the separate components of the model and their applicability to heterogeneous outcomes. In a world of incomplete etiologic concepts, imperfect measures of these incomplete etiologic concepts, and unknown boundaries between and within diagnostic categories, the situation is quite different. And this, as we have attempted to illustrate, is our present world. As our discussion has shown, there is a clear and consistent danger of missing vital aspects of the concepts (e.g., particular qualities of life stress that are important for the disorder) and of generating spuriously affirmative findings (e.g., through contamination of concepts and measures), both of which preclude a meaningful analysis of interactions between diatheses, stressors, or other predisposing factors.

We suggest that the essence of the diathesis—stress model is its implications for interactive analysis. This is in contrast to the complementary tendency to think in extensive terms and comprehensive theories. Interaction implies a more restrained focus, an in-depth probing of associations between the components of the model, often multidirectional and transpiring over time. In contrast, comprehensive implies a broad view of possible correlates, often sweeping and possessing functionally interchangeable components, that can combine in nonspecific ways. The first approach suggests an intensive study of specific factors and their integration over time within focused, prospective studies. It describes developmental dynamics: specific processes. The second approach suggests extensive study of suspected etiologic influences and their respective contributions. It describes linear, additive, and atemporal associations: nonspecific arrangements. We suggest that both approaches have their place, but that they should be clearly distinguished with respect to their virtues and limitations. The challenge is to maintain a focus on the meaning of interactions between diatheses and environment, and ultimately among other contributing

factors, without falling into the looseness that such thinking can engender. In our present world of incomplete concepts and imperfect measures, it would seem that the intensive rather than comprehensive focus on concepts, measures, and interactions most closely exemplifies the spirit–and may help to realize the promise–of diathesis—stress concepts in psychopathology research.

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**1**

The concept of a diathesis is as old as early conceptualizations of disease within naturalistic, or physical, origins. Derived from ancient Greek, the term can be traced at least to the writings of Galen (131—201, A. D. ) in his interpretations of Hippocratic theories of disease ( Entralgo, 1955 ; Simpson & Weiner, 1989 ). Notions of constitutional predisposition also dominate early theories of depression (or melancholia; Burton, 1621/1977 ; Jackson, 1986 ; Klibansky, Panofsky, & Saxl, 1979 ) and more generally of "nervous disease" ( López Piñero, 1983 ; Whytt, 1765 ). For present purposes, our definition is consonant with that provided in 1883 by Stearns's chapter "The Insane Diathesis": "a nervous system so sensitively constituted, and illy adjusted to its surroundings, that when brought in contact with unusually exciting influences, there may occur deranged instead of natural mental action, and it becomes more or less continuous instead of evenascent" (cited in Grob, 1983 , p. 40).

**2**

Although several research groups have adopted interviewer-based procedures (e.g., Alloy et al., 1988 ; Brown & Harris, 1978 ; Hammen et al., 1989 ), the actual guidelines, decision rules, and operational criteria are not typically explicit. This is not a trivial concern and unfortunately is a commonly ignored issue. An illuminating exception to this is the extensive and detailed work of Brown and Harris (1989) in the development of the Beford College Life Events and Difficulties Schedule (LEDS) and rating system. A detailed semi-structured interview with elaborate manuals provides extensive rules and criteria for defining events, distinguishing between related events, and differentiating events and difficulties. Listed are some 800 categories of experiences and over 5,000 case vignettes for standardizing stress definitions and ratings. These manuals are for major life events and difficulties and do not encompass the more common daily hassles or minor events. To include such stressors and thereby study stress comprehensively, adequate technologies for defining these latter experiences and for distinguishing them from major events and chronic stressors need to be developed (see also B. S. Dohrenwend et al., 1984 ). As suggested by the labor-intensive LEDS procedures for defining major events and difficulties, this would be an extremely demanding undertaking.

**3**

Because we are not concerned with the estimation of parameters, only with the properties of interactions for illustration purposes, we have omitted error terms from the model.

**4**

The existence of a threshold is currently a debatable and ultimately an empirical issue. Certainly not all diathesis—stress theories posit a threshold for the

diathesis (e.g., Abramson, Metalsky, & Alloy, 1989 ). However, many biological theories of depression emphasize the consistency of genetic/familial findings for depression, and discussions of these findings often invoke biological diatheses and genetic origins as clearly primary determinants of who develops the disorder (e.g., Andreasen, 1987 ; Clayton, 1986 ; Guze, 1989 ; Heston, 1988 ; Whybrow, Akiskal, & McKinney, 1984 ). As we see later in the article, the notion of a threshold also may be useful with respect to conceptualizing stress. Irrespective of the ultimate validity of thresholds, their inclusion in the present analysis is useful for simplifying issues pertaining to diathesis—stress interactions and for setting the foundation on which we develop more elaborate representations consonant with theories adopting a continuous view of the diathesis.

**5**

Parts of the next discussion are adapted from Cleary and Kessler (1982) . For simplicity, we have again omitted error terms from the equations.

**6**

The possibility of multiple diatheses raises other intriguing questions about associations between separate propensities to disorder and about associations of different diatheses with different forms of stress. For example, do different diatheses create additive or interactive risk for depression or contribute independently to separate subtypes of the disorder? Do different diatheses require different forms of stress for activation? Although our focus in the present article is on the manner in which diathesis—stress models can be informed by a conceptual analysis of stress theory and research, the complementary task of informing these models by a conceptual analysis of different domains of predisposition and their interactive influences could yield other valuable guidelines for theory and research.

**7**

By this we do not mean to imply that biological or genetic factors are not of importance in the genesis of depression or of possible subtypes of the disorder. It might be useful to think of diatheses in two senses with regard to this issue: one an active sense and one a passive (or permissive) sense. As we have noted before, our argument is based on the broad class of depressions and does not address hypothetical subtypes or differences in diatheses. Consequently, we cannot infer backwards from population estimates for depression about the frequency of specific active diatheses. However, we can infer backwards from such evidence concerning general passive diatheses. In other words, even if there are multiple subtypes of depression with separate diathetic factors (e.g., biological vs. cognitive), many people must still possess the "permissive" biologic diathesis for incurring depression.

**8**

The time periods over which life stress was assessed for these studies were 3 months (one study), 6 months (one study), 10 months (one study), 12 months (six studies). If studies assessing both events and difficulties that are based on a common 12-month time period alone are examined, the comparable figures are that an average of 83% cases experienced prior severe events or difficulties as compared with 33% of noncases.

**9**

Only 7 of the 11 cited studies included noncase comparison groups.

**10**

The data presented in Table 1 are adapted and rearranged from Brown and Harris (1978, p. 108) . Only severe events unrelated to each other are included

(events that do not arise from a common source, such as a serious marital argument, subsequent separation, and initiation of divorce proceedings). Note also that we do not include ongoing difficulties in this discussion. Whereas this does not affect the interpretation of the present issue involving additivity of events, it could be misleading with respect to more general issues of stress (i.e., events or difficulties) preceding depression (i.e., greater proportions of women had some form of stress before onset than is apparent from analyzing events alone; see Brown & Harris, 1986 ). Finally, for brevity, community cases and patients from this research are combined. Similar effects hold in regard to the additive potency of multiple severe events across both community cases of depression and the patient sample.

**11**

Note that more recent advances in clarifying the characteristics of stressors have led to even greater precision in predicting the onset of depression in the face of psychosocial adversity (e.g., approximating 50%; see Brown & Harris, 1989 ). These data suggest that it may be the matching of specific types of events with specific types of psychosocial vulnerability that is especially important in bringing about depression (see also Abramson, Metalsky, & Alloy, 1989 ). Although such findings may argue against the idea of above-threshold additivity (because the instances of multiple events may represent the greater probability of a match occurring rather than additivity per se), they further support the general conclusions of this argument: (a) There may be very specific qualities of environmental adversity that can lead to depression for specific people, (b) under such conditions, many people (if not all) have the capacity to become depressed, and (c) the incidence of such specific pathogenic matching in the general population (i.e., stressful conditions that precipitate depression) may be even less common than the estimates above suggest.

**12**

This argument assumes, we believe reasonably, a positively skewed distribution of the diathesis (i.e., more people with lower loadings). It also depends in part on the percentage of people with life stress who develop depression. The stronger the link between severe events and subsequent depression, the more persuasive is this line of reasoning (i.e., a lesser link leaves greater room for different loadings of the diathesis to determine who does and does not break down). Given recent estimates of approximately 50% of people developing depression with particular constellations of prior life stress ( Brown & Harris, 1989 ), the implications of this point for understanding the relative importance of diatheses merit consideration.

**13**

The actual degree to which this shift occurs is again a function of several factors, including the strength of diathetic influence on stress, the changes in the strength of this influence at differing potencies of the diathesis, the frequency of the differing potencies of the diathesis in the population, the incidence of stress that is not a consequence of the diathesis, and the impact of stress at the different potencies of the diathesis. Without more specific data on these matters, one can only conclude that to an unspecified degree, the high-risk people become more representative of depressed people.

**14**

Note that this suggests some degree of dependency between a diathesis for depression and these forms of life stress. It also suggests that both the diathesis and stress are important in the predictive scheme but leaves open the question of which dependency model cited previously is most appropriate (Model 1, in Figure 5 , or Model 3, in Figure 7 ).